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Association Between Glu298Asp Polymorphism of the eNOS Gene and Coronary No-Reflow in Patients Undergoing Primary Percutenous Intervention

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Objectıve: To assess whether Glu298Asp polymorphism of the endothelial nitric oxide sytnhase (eNOS) gene is associated with the occurrence of coronary no-reflow phenomenon.
Background: Genetic variants of endothelial nitric oxide synthase (eNOS) could influence individual susceptibility to coronary artery disease. Nitric oxide gene polymorphisms can effect both production and functions of nitric oxide and therfore may cause endothelial dysfunction. We hypothesized that Glu298Asp polymorphism of the eNOS gene may be associated with coronary no-reflow devolopment in patients with ST elevation myocardial infarction (STEMI) undergoing primary percutenous intervention.
Materials: We included 121 patients with STEMI that underwent primary percutenous coronary intervention. Of 121 patients 57 patients had coronary no-reflow and 64 patients were without coronary no-reflow. Coronary noreflow was defined as Thrombolysis In Myocardial Infarction flow grade 2 or less after intervention. Genotype was determined by polymerase chain reaction.
Results: Patient and control groups were simillar in terms of sex, age, diabetes, body mass index, hypertension and family history. Gensini and Syntax scores were lower in control group (47.13±24.8 vs 67.4±26.85 and 17.94±8.03 vs 25.48±10.3 p=0.001 p=0.001 respectively).In admission serum creatinine and blood glucose levels were lower in control group (0.84±0.17 vs 0.93±0.32 and 144.87±74.78 vs 177.16±94.42 p=0.004, p=0.0041 respectively). Initial and peak troponin levels were also lower in control group (0.25 [0.04-2.82] vs 1.55 [0.15-8.87] and 3.15 [1.28-8.52] vs 10 [3.6-25] p=0.025, p=0.0001 respectively). Neutrophil count and neutrophil to lymphocyte ratio was higher in noreflow group (8754.76±3428.08 vs10325.18±3950.56 and 5 [2.28-6.82] vs 7 [4.14-9.81] p=0.0025, p=0.001 respectively). The genotype frequencies of Glu298Asp polymorphism in control subjects were 59.38% for Glu/Glu, 39.06% for Glu/Asp, and 1.56% for Asp/Asp. On the other hand, in no-reflow patients, the genotype frequencies were 40.38% for Glu/Glu, 48.08% for Glu/Asp, and 11.54% for Asp/Asp. There was no statistically significant difference in terms of Glu/Glu and Glu/Asp polymorphisms between two groups. The proportion of Asp298 homozygotes was 11.54% in the no-reflow group and 1.56% in control group (p=0.032). In comparison with Glu/Glu genotype, the odds ratio (OR) for no-reflow occurence associated with the Asp/Asp genotype was 10.86 (1.22-96.39)
Conclusıon: In our study we found a significant association between Glu298Asp polymorphism of the eNOS gene and coronary noreflow phenomenon. Nitric oxide is one of the mediators that regulates endothelial functions and vascular tone. Polymorphisms at eNOS gene may adversely effect both production and function of nitric oxide. Coronary no-reflow phenomenon has multipl mechansims. Endothelial dysfunction and vasular spasm are also accused for no-reflow devolopment.

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QT Dispersion in the Severe Aortic Stenosis, before and after Transaortic Valve Implantation

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Background and Aım: In severe aortic stenosis (AS), electrophysiological changes are observed following mechanical stretch due to pressure overload. This electrical instability observes as development of after depolarization and dispersion of repolarization. The aim of this study was evaluated the ventricular repolarization changes following transcatheter valve implantation (TAVI).
Materıal-Methods: A prospective analysis of echocardiography and electrocardiography was evaluated before TAVI, same day and 1 week later. Ventricular repolarization was assessed with QT, QTc and in terms of dispersion across the myocardium; QT, QTc dispersion (QTD, QTDc). Twelve lead electrocardiography (ECG) was recorded for each subjects at a rate of 25 mm/s and 10 mm/V gain. The QT intervals were taken to be from the onset of the QRS to the end of the T wave. If U waves were present, the QT interval was measured to the nadir of the curve between the T and U waves. The measurements of the QT duration were performed manually by two of the investigators. To improve accuracy, measurements were performed with calipers and magnifying lens for defining the ECG deflection.
Results: 70 consecutive patients (male/female:19/51, age: 77,6 years, AS max/mean gradient: 85,2/52,8 mmHg, left ventricular ejection fraction:54,8%), diagnosed inoperable/high surgical risk severe aortic stenosis (logistic euroscore:%21,7, STS:7.7), were included the study. Before TAVI, QT dispersion and QTc dispersion were correlated with mean aortic valve area (p<0.001). There was statistically significant difference in QT dispersion and QTc dispersion (p<0.001). Patients' QTD, QTDc after TAVI (73.6±25.7, 74.8±29,4 respectively; QTD p: 0.044, QTDc p:0.030) and 1st week after TAVI (63.8±27.7, 66.1±30.6 respectively; QTD p<0.001, QTDc p<0.001) was significantly lower than before the TAVI (80.8±25.9, 83,3±26,4 respectively). Also statistically significant difference of QTD, QTDc were observed between after TAVI and 1st week after TAVI (QTD p:0.003, QTDc p:0.014).
Conclusıon: Sudden cardiac death (SCD) is major causes of death in patients with severe aortic stenosis, and also ventricular tachycardia, ventricular fibrillation are major causes of SCD. Decrease mechanical stretch due to pressure overload after TAVI, can also reduction across the myocardium of the dispersion of ventricular repolarization and could be risk reduction in patients with significant symptomatic aortic stenosis.

Graphic: QTD and QTDc changing with TAVI.

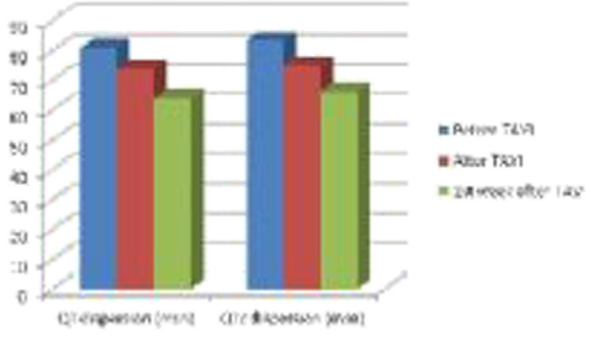

Table: Basal characteristics and procedural features

Characteristics	All Patients (70)
Male/Female (n)	19/51
Age (years)	77,6±6,7
BMI (kg/m²)	27,6±8,9
NYHA class II (n)	7
NYHA III (n)	47
NYHA IV (n)	16
STS	7,7±5,1
EuroScore (%)	21,7±15,9
Echocardiography parameters	
Maximal Gradient (mmHg)	85,2±18,6
Mean Gradient (mmHg)	52,8±13,3
LVEF (%)	54,5±14
AVA (cm²)	0,65±0,17
Procedural features	
Valve size mm (n)	
- 23	44
- 26	25
- 29	1
Discharging period after treatment (day)	5,5